Topical fluorides in caries prevention and management:

A North American perspective

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ABSTRACT

A review of evidence-based literature indicates incomplete evidence for the efficacy of most measures currently used for caries prevention, with the exception of fluoride varnishes and the use of fluoride-based interventions in the management of patients with hyposalivation. Not all fluoride agents and treatments are equal. Different fluoride compounds, different vehicles, and vastly different concentrations of fluoride have been used with different frequencies and durations of application. These variables can influence the clinical outcome with respect to caries prevention and management. The efficacy of topical fluoride in caries prevention depends on (a) the concentration of fluoride used, (b) the frequency and the duration of application, and, to a certain extent, (c) the specific fluoride compound used. The more concentrated the fluoride and the greater the frequency of application, the greater the caries reduction. Factors besides efficacy, such as practicality, cost and compliance, influence the clinician's choice of preventive therapy. For non-cavitated smooth surface carious lesions in a moderate caries-risk patient, the appropriate fluoride regimen would be semiannual professional topical application of a fluoride varnish containing 5% NaF (22,600 ppm of fluoride). In addition, the patient should use twice or thrice daily for at least 1 minute a fluoridated dentifrice containing NaF, MFP or SnF_2 (1,000-1,500 ppm of fluoride), and once daily for 1 minute a fluoride mouthrinse containing .05% NaF (230 ppm of fluoride). If the non-cavitated carious lesion involves a pit or fissure, the application of an occlusal

sealant would be the most appropriate preventive therapy. The management of the high caries-risk patient requires the use of several preventive interventions and behavioral modification, besides the use of topical fluorides. For children over 6 years of age and adults, both office and self-applied topical fluoride treatments are recommended. For office fluoride therapy at the initial visit, a high-concentration agent, either a 1.23% F APF gel (12,300 ppm of fluoride) for 4 minutes in a tray or a 5% NaF varnish (22,600 ppm of fluoride) should be applied directly to the teeth four times per year. Self-applied fluoride therapy should consist of the daily 5-minute application of 1.1% NaF or APF gel (5,000 ppm of fluoride) in a custom-fitted tray. For those who cannot tolerate a tray delivery owing to gagging or nausea, a daily 0.05% NaF rinse (230 ppm of fluoride) for 1 minute is a less effective alternative. In addition, the patient should use twice or thrice daily for at least 1 minute a fluoridated dentifrice as described above for treatment of non-cavitated carious lesions. In order to avoid unintentional ingestion and the risk of fluorosis in children under 6 years of age, fluoride rinses and gels should not be used at home. Furthermore, when using a fluoride dentifrice, such children should apply only a pea-size portion on the brush, should be instructed not to eat or swallow the paste, and should expectorate thoroughly after brushing. Toothbrushing should be done under parental supervision. To avoid etching of porcelain crowns and facings, neutral NaF is indicated in preference to APF gels for those patients who have such restorations and are applying the gel daily. The rationale for these recommendations is discussed. Important deficiencies in our knowledge that require further research on topical fluoride therapy in populations with specific needs are identified.

KEY WORDS: topical fluoride, fluoride dentifrice, fluoride mouthrinse, fluoride varnish, caries risk, hyposalivation, clinical trials, caries prevention.

INTRODUCTION

Before starting my presentation on the role of topical fluorides in caries prevention and management of high-risk patients, let me say that water fluoridation must be an important part of any oral health preventive program. The benefits are irrefutable, ranging from 11 to 40% less decay, depending on dentition, population, and use of preventive practices (1). Because of the diffusion effect of fluoride from foods and beverage products processed in optimally fluoridated areas being consumed in "non-fluoridated" areas, this may be an underestimation of the total benefit of water fluoridation, especially in high diffusion areas (2). I am well aware of the mantra, repeated *ad nauseum,* that water fluoridation acts primarily topically (3-6), but simply repeating it does not make it correct. Findings from carefully conducted epidemiological studies on humans, not simply *in vitro* models, show that water fluoridation provides both pre- and post-eruptive benefits, that is both systemic and topical in combination (7, 8). Studies in the Netherlands, based on time of exposure to fluoride (pre- and/or post-eruptive), indicate that fluoride has an important pre-eruptive effect on caries in permanent teeth (7). Approximal smooth surface caries reduction was attributable to 50% pre- and 50% post-eruptive fluoride exposure, whereas pit and fissure reduction was attributable to 66% pre- and 33% post-eruptive fluoride exposure. On free smooth surfaces, caries reduction was due to 25% pre- and 75% post eruptive exposure to fluoride. Recent Australian studies (8) have confirmed these earlier findings, namely that children with optimal exposure to fluoridated water, both pre- and post-eruptively, had the lowest caries on all surface types. These findings support the policy of water fluoridation as a public health measure for the prevention of caries.

Two of the questions addressed in the review of the literature by Bader et al.(9) are the effectiveness of strategies to arrest or reverse the progress of initial lesions before they become cavitated, and the most effective interventions to mitigate the caries attack in persons identified as being at high risk of caries. The authors limited themselves to the consideration of reports concerning methods applied in a professional setting and, in evaluating management of high caries-risk patients, to only those studies where the subjects had been thus identified, based on past caries experience and/or bacteriological testing. They did not include hyposalivation as a criterion of prediction of high caries risk, perhaps because there is considerable variability in what constitutes normal salivary flow rate (10, 11). There is a broad consensus that individuals with severe salivary gland dysfunction are indeed at high risk of developing caries (12-14). However, the exact cut-off with respect to what constitutes a low salivary flow rate varies throughout the literature, such as < 0.7 ml/min for whole stimulated saliva and 0.10 to 0.16 ml/min for whole unstimulated saliva (15-19). There is no consensus about a flow rate value that is “abnormal”, although recently it has been suggested that a 45% reduction in a person’s stimulated flow rate could be considered an objective measure of salivary hypofunction (11). Measuring salivary flow rates is not routinely performed in dental offices, can be time-consuming if the patient has hyposalivation, and may be difficult to standardize when done only occasionally. Nevertheless, monitoring secretory function longitudinally can be incorporated by dentists as a routine examination procedure and can be a valuable adjunct to oral diagnosis (10).

With respect to the management of non-cavitated carious lesions, the review (9) identified only five studies that met the evidence-based criteria, the evidence was rated incomplete, and no

specific intervention was identified as effective. In the management of high caries-risk patients, 22 studies were considered, of which nine involved fluoride interventions and six involved a combination of treatments, some including fluoride. It was concluded that the evidence supporting the use of fluoride varnishes in caries prevention for such patients was fair, while other fluoride therapeutic agents showed non-significant small protective benefits, but the data were incomplete.

Similarly, in a review of seven studies of patients with hyposalivation due to therapeutic irradiation for head and neck cancer Newbrun (20) found that the treatment protocols employed had all used self-applied topical fluoride agents, sometimes in combination with chlorhexidine, but had varied considerably in the type (stannous fluoride {SnF_2}, sodium fluoride {NaF} or acidulated phosphate fluoride {APF}), concentration (230 to 12,300 ppm F), duration and frequency of the fluoride agent used (1 minute once per day to 5 minutes twice per day). In addition, some protocols had included professional applications of topical fluoride. The form of self-application had also varied, from rinsing to brushing-on of gels, to the use of custom-fabricated individual mouth trays. Most of these studies had used small numbers of patients and had not had a true control group for obvious ethical reasons; moreover, when the studies had compared different treatments, the assignment of patients to treatment groups was not always random. Most patients had not been studied for a minimum of two years. The extent of patient compliance with the daily recommended regimens had varied, doubtlessly because of differences in the populations as well as in the practicality and acceptability of the regimens. In addition, the data had not always been expressed in the conventional DMFT/S form. For all these

reasons, Newbrun was unable to draw firm conclusions, based on those studies, as to the best protocol to recommend for patients with hyposalivation.

DISCUSSION

Findings from extensive clinical trials, mostly in children, on the efficacy of fluoride dentifrices and/or other self-applied fluoride agents as well as on professionally applied (office or school) fluoride treatments, not included in the evidence-based report (9), are relevant to strategies to arrest or reverse the progress of initial lesions and the most effective interventions in treating persons identified as being at high caries risk.

The first point, which would seem to be self-evident, is that not all fluoride agents and treatments are equal. Different fluoride compounds, different vehicles, and vastly different concentrations of fluoride have been used with different frequency and duration of application (Table 1). All of these variables can influence the clinical outcome with respect to caries prevention and management. The efficacy of topical fluoride in caries prevention depends on (a) the concentration of fluoride used, (b) the frequency with which it is applied and probably also the duration of application, and, to some extent, (c) the specific fluoride compound used (21, 22).

a. With regard to the concentration of fluoride used, the preponderance of fluoride dentifrice studies have shown a dose/response effect (21, 23), and the trend in clinical effectiveness of professionally applied topical fluoride agents is similar (24-26) (Table 2).

b. With respect to the frequency of topical fluoride application, in studies using the same commercial stannous fluoride dentifrice, the efficacy of unsupervised once per day or *ad libitum*

use was about a 21% caries reduction (27, 28), whereas the efficay of supervised thrice per day use was about a 45% caries reduction (29, 30) (Table 3). Similarly, Marthaler (31) found that in studies using more concentrated fluoride brush-on gels or solutions, those that employed 15 or more applications per year had 40 to 50% caries reductions, whereas those with four to five applications per year were less efficacious. Although a recent report (32) failed to demonstrate an association between frequency of use of professionally applied topical fluoride and the placement of interproximal restorations, the reason may well be that dentists' propensities to place restorations in children overwhelmed any preventive effect of the topical fluoride applications. Furthermore, this analysis of dental insurance claims in relation to professional topical fluoride application assumed that all applications were made according to "standard practice" and, therefore, had the same efficacy (32).

There are not any controlled clinical trials in which the same concentration of a topical fluoride agent has been tested for varying durations of application. However, in vitro testing of both NaF and APF solutions has shown that fluoride uptake is time related, and in the case of APF solutions the most rapid uptake occurs during the first 4 minutes (33). Although some manufacturers claim that their brand of gel or foam will give provide near maximum uptake in 1 minute, these products lack documented testing for clinical efficacy. Until such data are forthcoming, the recommended duration of application of APF agents should be 4 minutes. One advantage of fluoride varnishes is that they adhere to tooth surfaces, thereby permitting prolonged fluoride exposure and uptake. In a meta-analysis of the efficacy of fluoride varnishes by Helfenstein and Steiner (25), using very rigid criteria for inclusion of data (only test vs. control, no split mouth trials; only studies with two applications per year; sample size, mean

values and standard deviations had to be given), eight studies fulfilled these criteria. They found a mean caries reduction of 38% for fluoride varnishes. Similarly, in their comprehensive review of evidence-based literature, Bader et al. (9) reported incomplete evidence for the efficacy of most measures currently used for caries prevention, with the exception of fluoride varnishes.

Other studies were performed quite a long time ago and may not meet contemporary criteria for inclusion in an evidence-based review. Nevertheless, there is a consistency of results across studies, namely that the more concentrated the fluoride and the greater the frequency of application, the greater the caries reduction. The fact that topical treatments differ in efficacy is often ignored by some who simply refer to "topical fluoride treatment" as if all such treatments were equivalent (32, 34). Of course, other factors besides efficacy, such as safety, practicality, cost and compliance, influence the clinician's choice of preventive therapy.

RECOMMENDATIONS FOR THERAPY

Treatment of non-cavitated carious lesions

Let us assume (a) that a non-cavitated carious lesion occurs in a moderate caries-risk patient and (b) that it involves a smooth surface. The appropriate fluoride regimen would be semiannual professional topical application of a fluoride varnish, containing 5% NaF (22,600 ppm of fluoride), to the non-cavitated carious lesion. If the patient is to be treated more than twice per year, then an application of a fluoride varnish should be done at each visit. In addition, the patient should use twice or thrice daily for at least 1 minute a fluoridated dentifrice containing NaF, MFP or SnF_2 (1,000-1,500 ppm of fluoride), and once daily for 1 minute a fluoride mouthrinse containing 0.05% NaF (230 ppm of

fluoride). Note that I am quite specific about the agents, the fluoride concentrations, and the frequency and duration of use because they all are important in the outcome. However, many clinicians think that if a carious lesion is detectable radiographically, even if the surface appears intact, it has already advanced beyond the radiolucency and therefore should be restored (35). Histologic and microradiographic findings indicate that caries is usually more advanced than it appears in clinical radiographs (36, 37). Others (38) suggest that if the enamel surface is intact, small lesions detected radiographically need not be restored immediately and that restoration can be delayed or avoided if adequate preventive procedures are carried out. Of course, any such non-cavitated carious lesions need to be monitored regularly to ensure that preventive treatment is effective and that the lesions are not progressing. Elderton (39) has proposed a decision tree for the clinical management of carious lesions in which caries is judged to be arrested, active, or unable to be judged. Only in the latter two instances are the criteria for restorative treatment fulfilled. However, I believe that one should not decide whether or not to restore on the basis of an individual tooth surface, but rather on the evaluation of the patient's caries risk as a whole.

Pit and fissure tooth surfaces require special assessment and management because diagnosis is less reliable at these sites. The application of sealants to early fissure lesions produces an immediate bactericidal effect (40) and causes a gross reduction in the number of viable bacteria in carious dentin beneath the sealant (41). If the non-cavitated carious lesion involves a pit or fissure, pre-eruptive fluoride exposure is more effective

than topical fluoride (7, 8). Accordingly the application of occlusal sealants would be the most appropriate preventive therapy once a tooth is erupted.

Management of high caries-risk patients

The management of a high caries-risk patient, whether child or adult, requires the use of several preventive interventions and behavioral modifications, not just the use of topical fluorides (20, 42, 43). For children over 6 years of age and adults, both office and self-applied topical fluoride treatments are recommended. For office fluoride therapy at the initial visit, a high-concentration agent, either a 1.23% F APF gel (12,300 ppm of fluoride) for 4 minutes in a tray or a 5% NaF (22,600 ppm of fluoride) varnish should be applied directly to the teeth. Since the application frequency is an important factor in determining efficacy, four such applications per year have been recommended (44). Self-applied fluoride therapy should consist of the daily 5-minute application of 1.1% NaF or APF gel (5,000 ppm of fluoride) in a custom-fitted tray. For those who cannot tolerate a tray delivery owing to gagging or nausea, a daily 0.05% NaF rinse (230 ppm of fluoride) for 1 minute is a less effective alternative. In addition, the patient should use twice or thrice daily for at least 1 minute a fluoridated dentifrice as described above for treatment of non-cavitated carious lesions. In order to reduce the risk of unintentional ingestion and fluorosis, for children under 6 years of age fluoride rinses and gels should not be used at home. Furthermore, when using a fluoride dentifrice, such children should apply only a pea-size portion on the brush, should be instructed not to eat or swallow the paste, and should expectorate thoroughly after brushing. Preferably toothbrushing should be under parental supervision. To avoid etching of porcelain crowns and facings, neutral NaF is indicated in preference to APF gels for those patients who have such restorations and are applying the gel on a daily basis (45).

RECOMMENDATIONS FOR RESEARCH

Non-cavitated carious lesions and patients diagnosed as being at high caries risk must be treated; accordingly, it is unethical to perform studies with placebo control groups. Many different fluoride agents (NaF, APF, SnF_2, organic fluorides and fluoride varnishes) have been tested for topical use in caries prevention, and the duration as well as the frequency of application have varied. Unfortunately, no head-to-head comparisons of regimens and agents have been published. At the Clinical Investigations and Patient Care Branch of NIDCR, the center with the largest number of patients with hyposalivation and therefore at high risk of caries, these agents were used interchangeably in managing the patients' dental problems, but their efficacy was not compared (46). This branch no longer exists as such, and the focus has changed; it is now the Gene Therapy and Therapeutics Branch of NIDCR. In order to obtain sufficient numbers of patients, it would be appropriate for NIDCR to issue a multicenter contract to test different fluoride agents and different durations and frequencies of application, with respect not only to relative effectiveness but also to cost, practicality, and acceptability in caries prevention and reversal. Such studies need to be conducted for a minimum of two years' duration and should record patient compliance and any deleterious side effects. The protocol should be specified and methods of application and evaluation standardized to ensure uniformity of the different center studies. Assignment of patients to different treatment modalities must be randomized and examiners blinded as to the treatment regimen. Appropriate statistical analyses must be performed. For high caries-risk patients, other agents besides fluoride

should be tested as adjuncts, and the best ways of behavioral modification to achieve good dietary and oral hygiene habits should be determined.

CONCLUDING COMMENTS

Although the evidence for the efficacy of fluoride interventions in the prevention and management of caries may be incomplete or only fair at best, the clinician must have some guidelines in treating patients at risk. Specific agents and methods of use must be recommended, but at present such recommendations may seem to be based on empirical clinical discernment. The recommendations listed above are my personal judgment-call. However, they are based on integration of data from extensive clinical trials, mostly in children, on the efficacy of fluoride dentifrices and/or other self-applied fluoride agents as well as on professionally applied (office or school) fluoride treatments.

REFERENCES

1. Newbrun E. Effectiveness of water fluoridation. J. Public Hlth. Dent. 1989;49:279-289.

2. Griffin SO, Gooch BF, Lockwood SA, Tomar SL. Quantifying the diffused benefit from water fluoridation in the United States. Community Dent Oral Epidemiol 2001;29:120-129.

3. Beltran ED, Burt BA. The pre- and posteruptive effects of fluoride on the caries decline. J. Public Hlth. Dent. 1988;48:233-240.

4. Thylstrup A. Clinical evidence of pre-eruptive effects of fluoride in caries prevention. J Dent Res 1990;69 (suppl):742-750.

5. Limeback H. A re-examination of the pre-eruptive and post-eruptive mechanism of the anti-caries effects of fluoride: is there any anti-caries benefit from swallowing fluoride? Community Dent Oral Epidemiol 1999;27:62-71.

6. Featherstone JDB. Prevention and reversal of dental caries: role of low level fluoride. Community Dent Oral Epidemiol 1999;27:31-40.

7. Groeneveld A, van Eck AAJM, Backer Dirks O. Fluoride in caries prevention: is the effect pre- or post-eruptive? J Dent Res 1990;69 (Spec Issue):751-755.

8. Singh KA, Spencer AJ, Armfield JM. Pre- and post-eruption fluoride exposure and caries by surface type. J Dent Res 2000;79:159.

9. Bader JD, Shugars DA, Rozier G, et al. Diagnosis and management of dental caries. Evidence Report. Chapel Hill: University of North Carolina, 2000.

10. Ship JA, Fox PC, Baum BJ. How much saliva is enough? J Am Dent Assoc 1991;122:63-69.

11. Ghezzi EM, Lange LA, Ship JA. Determination of variation of stimulated salivary flow rates. J Dent Res 2000;79:1874-1878.

12. Mandel ID. The role of saliva in maintaining oral homeostasis. J Am Dent Assoc 1989;119:298-304.

13. Atkinson JG, Baume BJ. Salivary enhancers. J Dent Educ 2001:this issue.

14. Leone CW, Oppenheim FG. Physical and chemical aspects of saliva as indicators of risk for dental caries. J Dent Educ 2001:this issue.

15. Ben-Aryeh H, Spielman A, Szargel R, Gutman D, Scharf J, Nahir M. Sialochemistry for diagnosis of Sjögren's syndrome in xerostomic patients. Oral Surg Oral Med Oral Pathol 1981;52:487-490.

16. Rask PI, Emilson CG, Krasse B, Sundberg H. Effect of preventive measures in 50-60-year-olds with a high risk of dental caries. Scand J Dent Res 1988;96:500-504.

17. Sreebny LM, Valdini A. Xerostomia. Part I: relationship to other symptoms and salivary gland hypofunction. Oral Surg Oral Med Oral Pathol 1988;66:451-458.

18. Spak CJ, Johnson G, Ekstrand J. Caries incidence, salivary flow and efficacy of fluoride gel treatment in irradiated patients. Caries Res 1994;28:388-393.

19. Navazesh M, Christensen C, Brightman V. Clinical criteria for the diagnosis of salivary hypofunction. J Dent Res 1992;71:1363-1369.

20. Newbrun E. Current treatment modalities of oral problems of patients with Sjögren's syndrome: caries prevention. Adv Dent Res 1996;10:29-34.

21. Newbrun E. Current regulations and recommendations concerning water fluoridation, fluoride supplements and topical fluoride agents. J Dent Res 1992;71(special issue):1255-1265.

22. Newbrun E. The evolution of professionally applied topical fluoride therapies. Comp Contin Educ Dent 1999;20:5-9.

23. Ripa LW. Clinical studies of high-potency fluoride dentifrices:a review. J Am Dent Assoc 1989;118:85-91.

24. Horowitz HS, Heifetz SB. Topically applied fluorides. In: Newbrun E, ed. Fluorides and Dental Caries. 3rd ed. Springfield: C.C.Thomas, 1986:71-114.

25. Helfenstein U, Steiner M. Fluoride varnishes (Duraphat): A meta-analysis. Community Dent Oral Epidemiol 1994;22:1-5.

26. van Rijkom HM, Truin GJ, van't Hof MA. A meta-analysis of clinical studies on the caries inhibiting effect of fluoride gel treatment. Caries Res 1998;32:83-92.

27. Jordan WA, Peterson JK. Caries-inhibiting value of a dentifrice containing stannous fluoride. Final report of a two-year study. J Am Dent Assoc 1959;58:42-46.

28. Horowitz HS, Law FE, Thompson MP, Chamberlin SR. Evaluation of a stannous fluoride dentifrice for use in dental public health programs. I. Basic findings. J Am Dent Assoc 1966;72:408-422.

29. Peffley GE, Muhler JC. The effect of a commercially available stannous fluoride dentifrice under controlled brushing habits on dental caries incidence in children: Preliminary report. J Dent Res 1960;39:871-875.

30. Bixler D, Muhler JC. Experimental clinical human caries test design and interpretation. J Am Dent Assoc 1962;65:482-490.

31. Marthaler T. Clinical cariostatic effects of various fluoride methods and programs. In: Ekstrand J, Fejerskov O, Silverstone LM, eds. Fluoride in dentistry. Copenhagen: Munksgaard, 1988:252-275.

32. Eklund SA, Pittman JL, Heller KE. Professionally applied topical fluoride and restorative care in insured children. J. Public Hlth. Dent. 2000;60:33-38.

33. Joyston-Bechal S, Duckworth R, Braden M. The mechanism of uptake of ^{18}F by enamel from sodium fluoride and acidulated phosphate fluoride solution labeled with ^{18}F. Arch Oral Biol 1973;18:1077-1089.

34. Sciubba JJ. Sjögren's syndrome: pathology, oral presentation, and dental management. Comp Contin Educ Dent 1994;15:1084-1095.

35. Christensen GJ. Initial carious lesions: when should they be restored? J Am Dent Assoc 2000;131:1760-1762.

36. Newbrun E, Brudevold F, Mermagen H. A microradiographic evaluation of occlusal fissures and grooves. J Am Dent Assoc 1959;58:26-31.

37. Gwinnett AJ. A comparison of proximal carious lesions as seen by clinical radiography, contact microradiography and light microscopy. J Am Dent Assoc 1971;83:1078-1080.

38. Silverstone LM, Johnson NW, Hardie JM, Williams RAD. Dental caries: aetiology, pathology, and prevention. London: Macmillan, 1981.

39. Elderton RJ. Principles of decision-making to achieve oral health. In: Ulrig U, ed. Professional prevention in dentistry. Baltimore: Williams and Wilkins, 1994:1-27.

40. Thielade E, Fejerskov O, Migasena K. Effect of fissure sealing on the microflora in occlusal fissures of human teeth. Arch Oral Biol 1977;22:251-259.

41. Jensen ØE, Handelman SL. Effect of autopolymerizing sealant on viability of microflora in occlusal dental sealants. Scand J Dent Res 1980;88:392-398.

42. Newbrun E. Preventing dental caries: current and prospective strategies. J Am Dent Assoc 1992;123:68-73.

43. Rozier RG. Effectiveness of methods for the primary prevention of dental caries: areview of the evidence. J Dent Educ 2001:this issue.

44. Council on Access Prevention and Interprofessional Relations. Caries diagnosis and risk assessment: a review of preventive strategies and management. J Am Dent Assoc 1995;126:1S-24S.

45. Council on Dental Materials Instruments and Equipment. Status report: effect of acidulated phosphate fluoride on porcelain and composite restorations. J Am Dent Assoc 1988;116:115.

46. Wright WE, Haller JM, Harlow SA, Pizzo PA. An oral disease prevention program for patients receiving radiation and chemotherapy. J Am Dent Assoc 1985;110:43-47.

Table 1. Range of therapeutic fluoride concentrations used to prevent caries

Method/vehicle	Fluoride concentration (ppm F)
Water supplies	0.7-1.2
Fluoridated salt	200-250
Mouthrinse, daily	230
Dentifrices, children	250-500
Mouthrinse, weekly	920
Dentifrices, adult	1,000-1,500
Self-applied gels or rinses, prescription	5,000
Professionally applied solutions (NaF)	9,200
Professionally applied solutions, gels, foams (APF)	12,300
Professionally applied solutions (SnF_2)	19,500
Professionally applied varnishes	22,600

Table 2. Comparative Effectiveness of Professionally Applied Topical Fluoride Agents

Agent	Fluoride concentration (ppm F)	Average effectiveness* % caries reduction
2% NaF	9,200	29
APF (1.2% F)	12,300	22
8% SnF_2	19,500	32
Fluoride varnish (5 % NaF)	22,600	38

*Source: Effectiveness estimates from Horowitz (24), van Rijkom (26) and Helfenstein (25).

Table 3. Frequency of supervised toothbrushing with a stannous fluoride dentifrice* and caries reduction

Study	Brushing frequency	Subject ages (years)	Study length	DMFS reduction
Jordan 1959 (27)	1x day	8-12	2 years	21%
Horowitz 1966 (28)	1x day + *ad libitum* home use	6-10	2 years	21%
Peffley 1960 (29)	3x day	10-15	2 years	46%
Bixler 1962 (30)	3x day	12-16	8 months	45%

*Crest ™